
Perinatology

Fetal Environment

- Fetus dependent on mare
  - Everything comes from mare
  - No communication of changing needs
- Insufficient resources delivered
  - Survival depends on redistribution of limited resources
    - Sophisticated compensatory responses
    - Response to challenges
- Fetus approaching term
  - Metabolic demands at limit of placenta's ability
  - Any disruption - devastating results

Threats to Fetal Well-being
Unique Aspects Placental Blood Flow

- Fetal Foal
  - Placental blood flow - 50% of the dam's flow
- Other Fetal Species
  - Placental blood flow - 75% of the dam's flow
- Fetal Foal under anesthesia
  - Dramatic decrease in fetal placental blood flow
    - 38% of maternal flow
  - No significant change in maternal placental blood flow

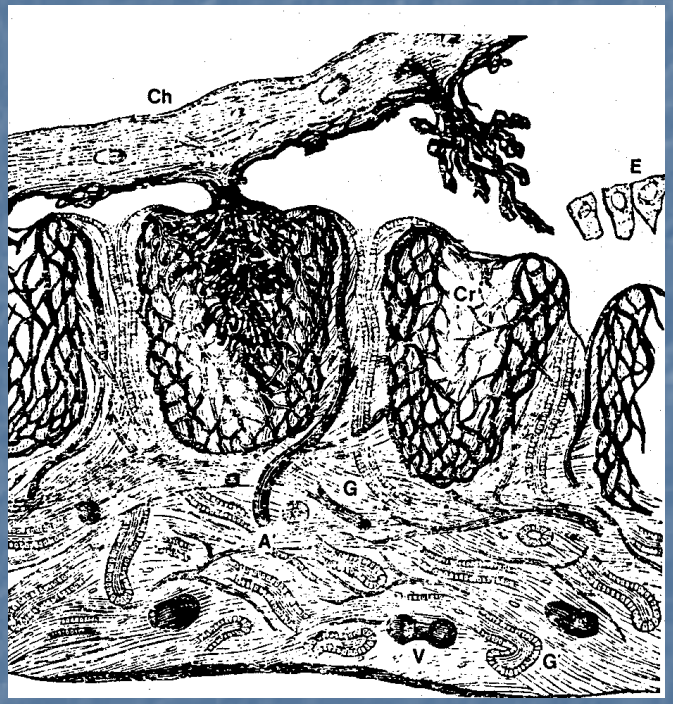
Fetal Resuscitation
Maintenance of Placental Perfusion

- Aggressively treat hypovolemia in dam
- Aggressively treat hypotension in the dam
- Avoid anesthesia in late term mares

Threats to Fetal Well-being Lack of O_2 Delivery

- Maternal threats
  - Maternal anemia
  - Maternal hypoxemia
  - Maternal decrease in perfusion
- Fetal response
  - Unique aspect of placentation
  - Placental oxygen transport mechanisms
  - Fetal physiologic adjustments

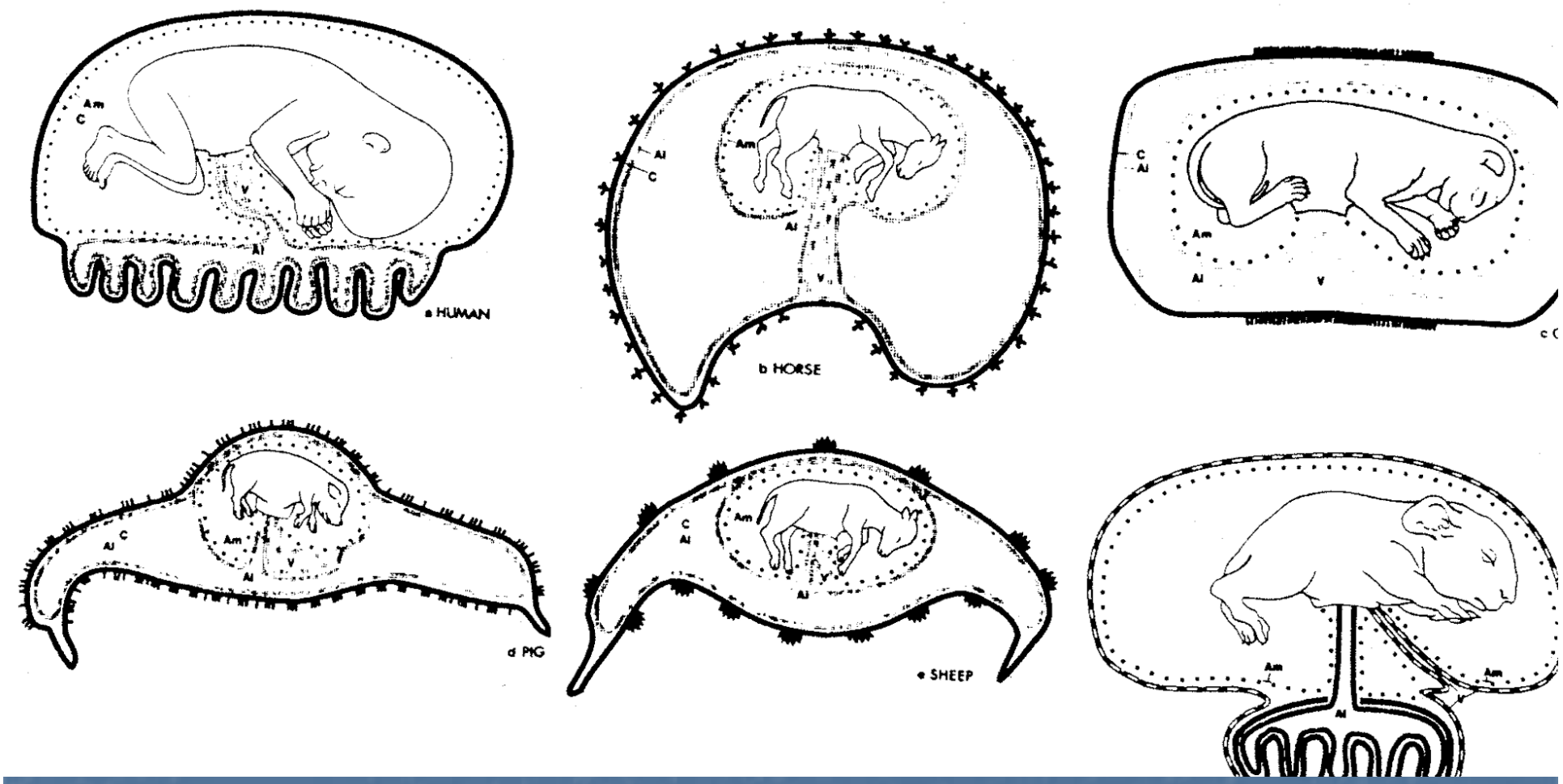
Historical Investigations of the Equine Placenta



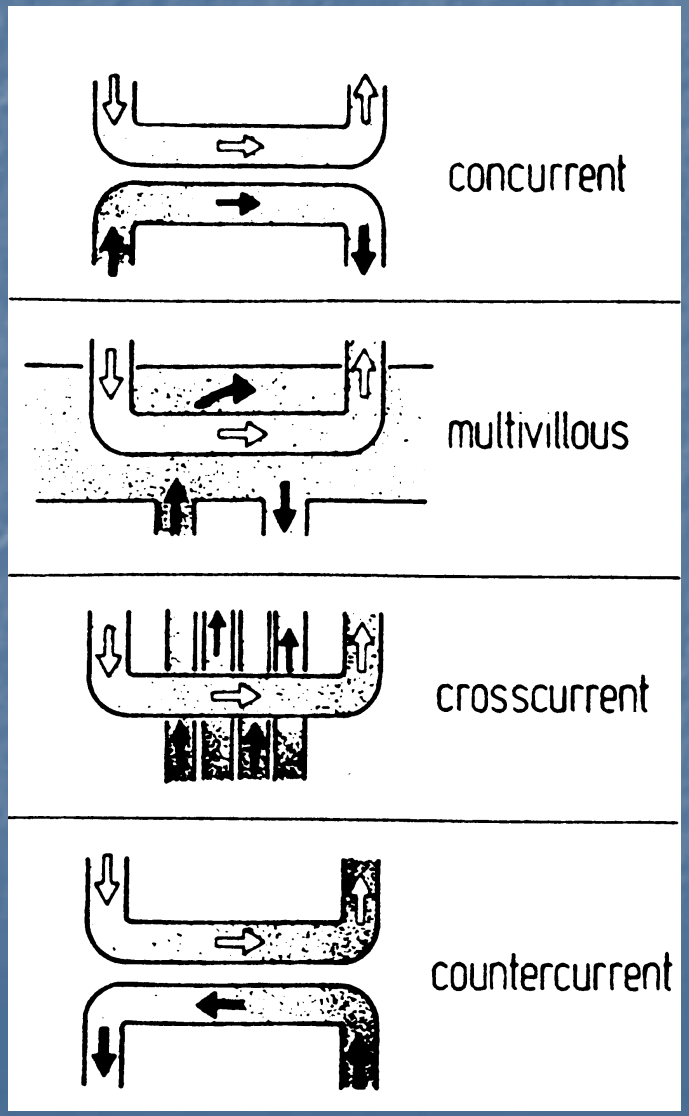
From: Turner (1876)
Lectures on the Comparative Anatomy of the Placenta

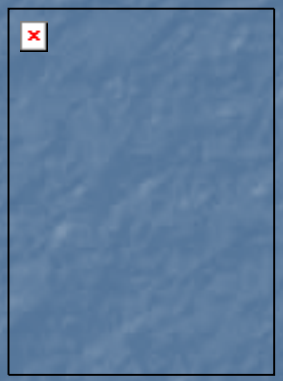
From: Ruini (1598) Anatomia del Cavallo

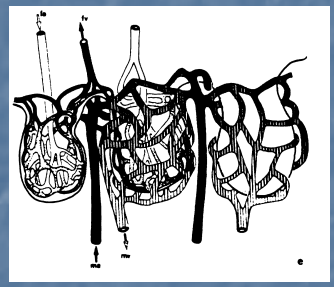
Placentation

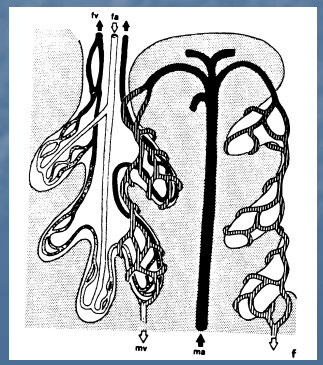


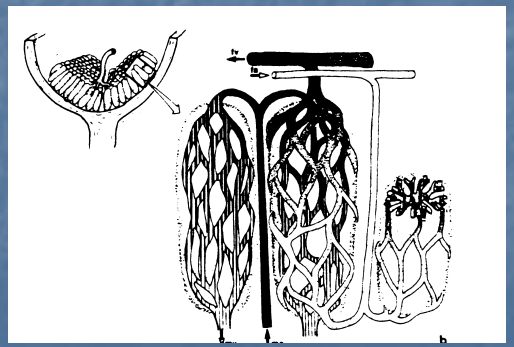
Placental Circulation



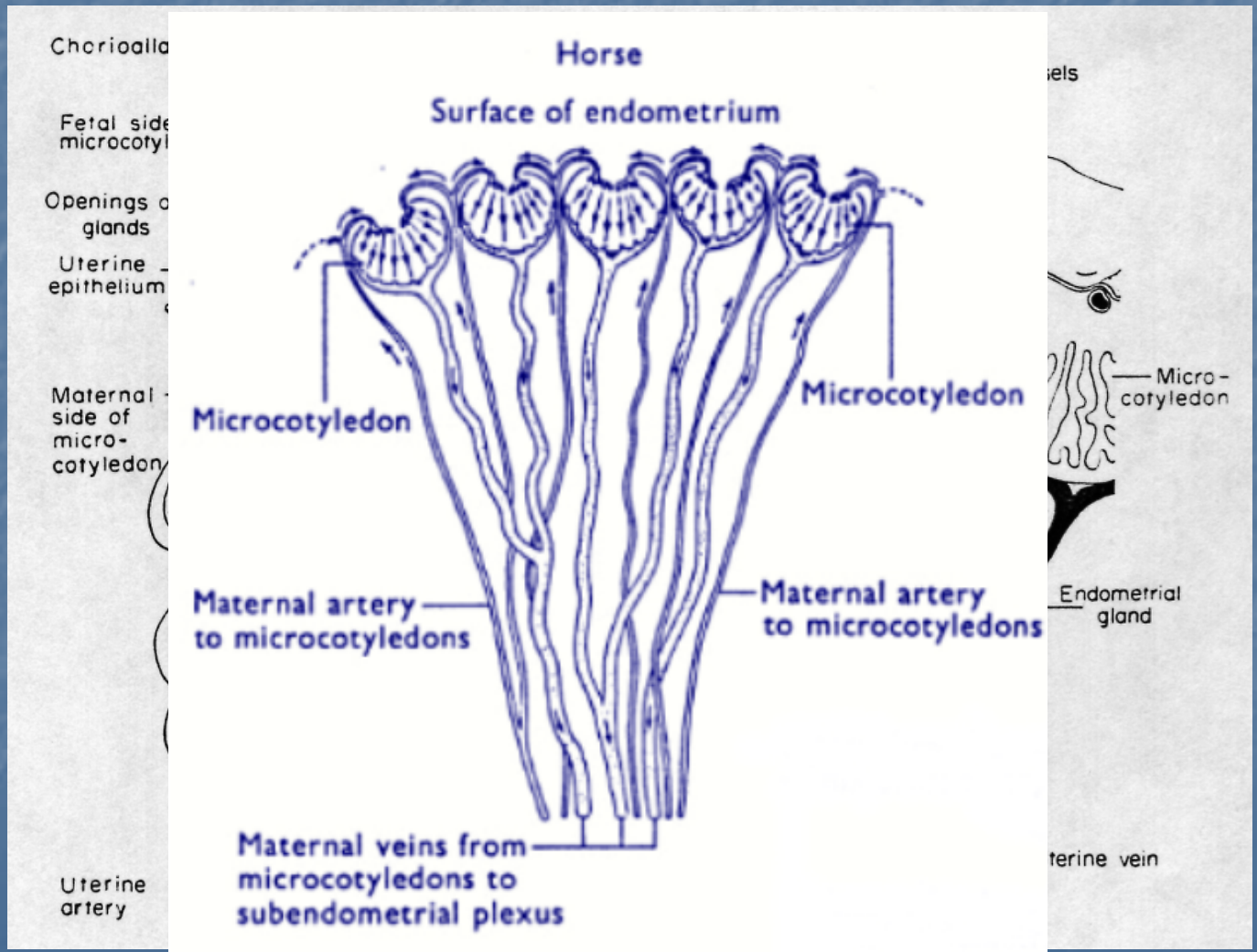




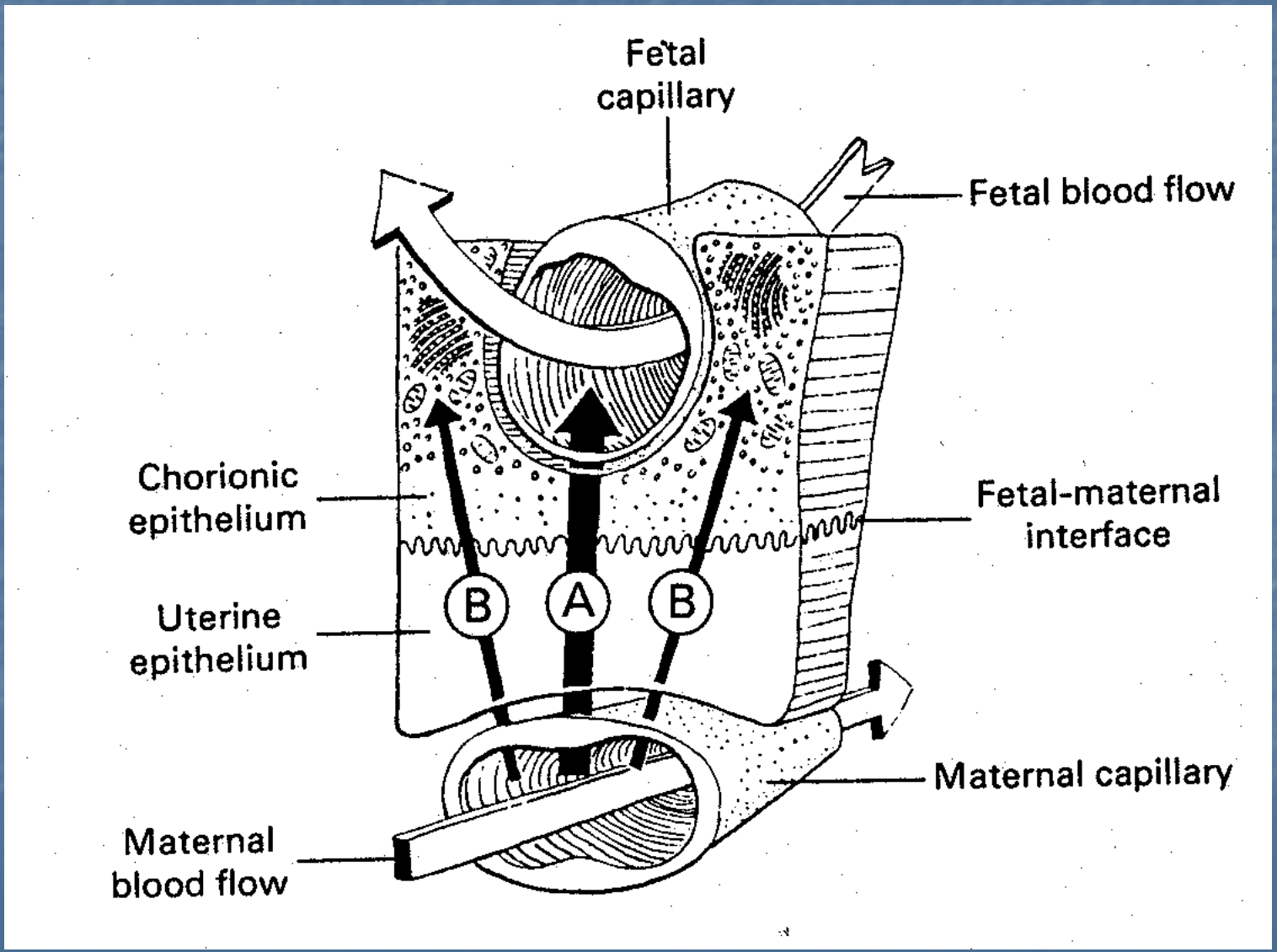




Equine Placentation



Countercurrent Circulation



From: Samuel 1976 J. Repro. Fert. 48:257

Effect of Maternal Oxygen Therapy

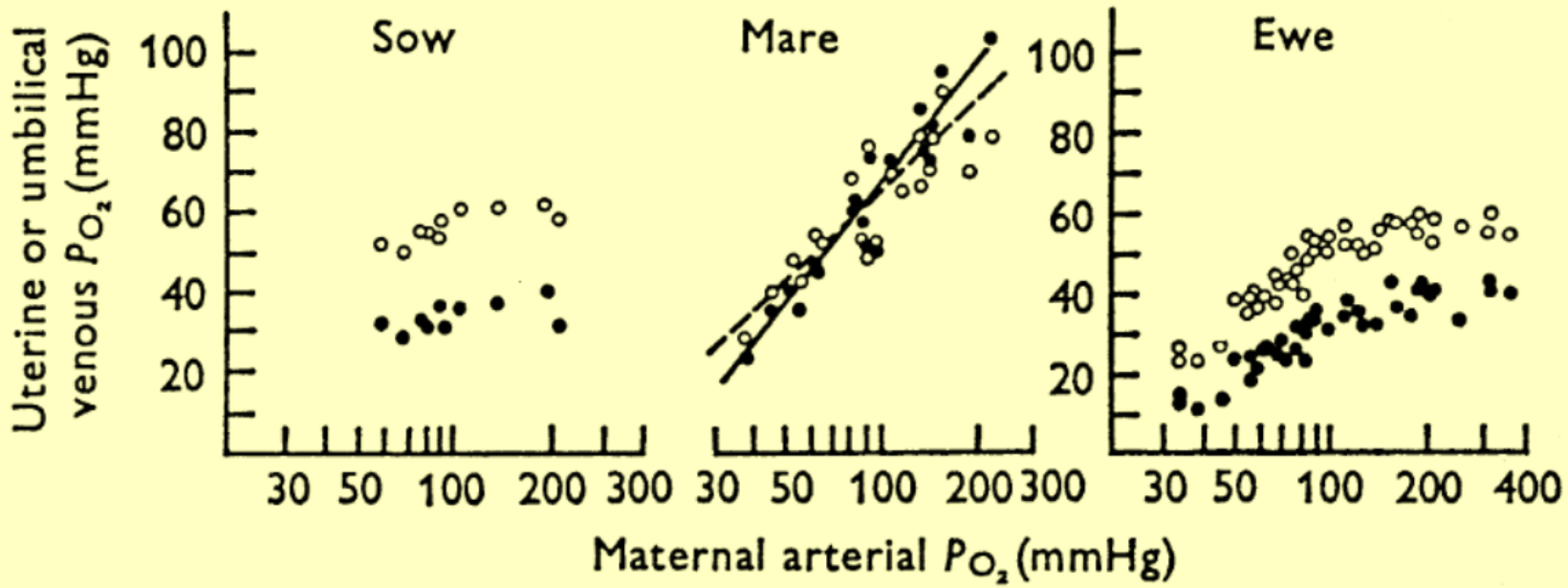
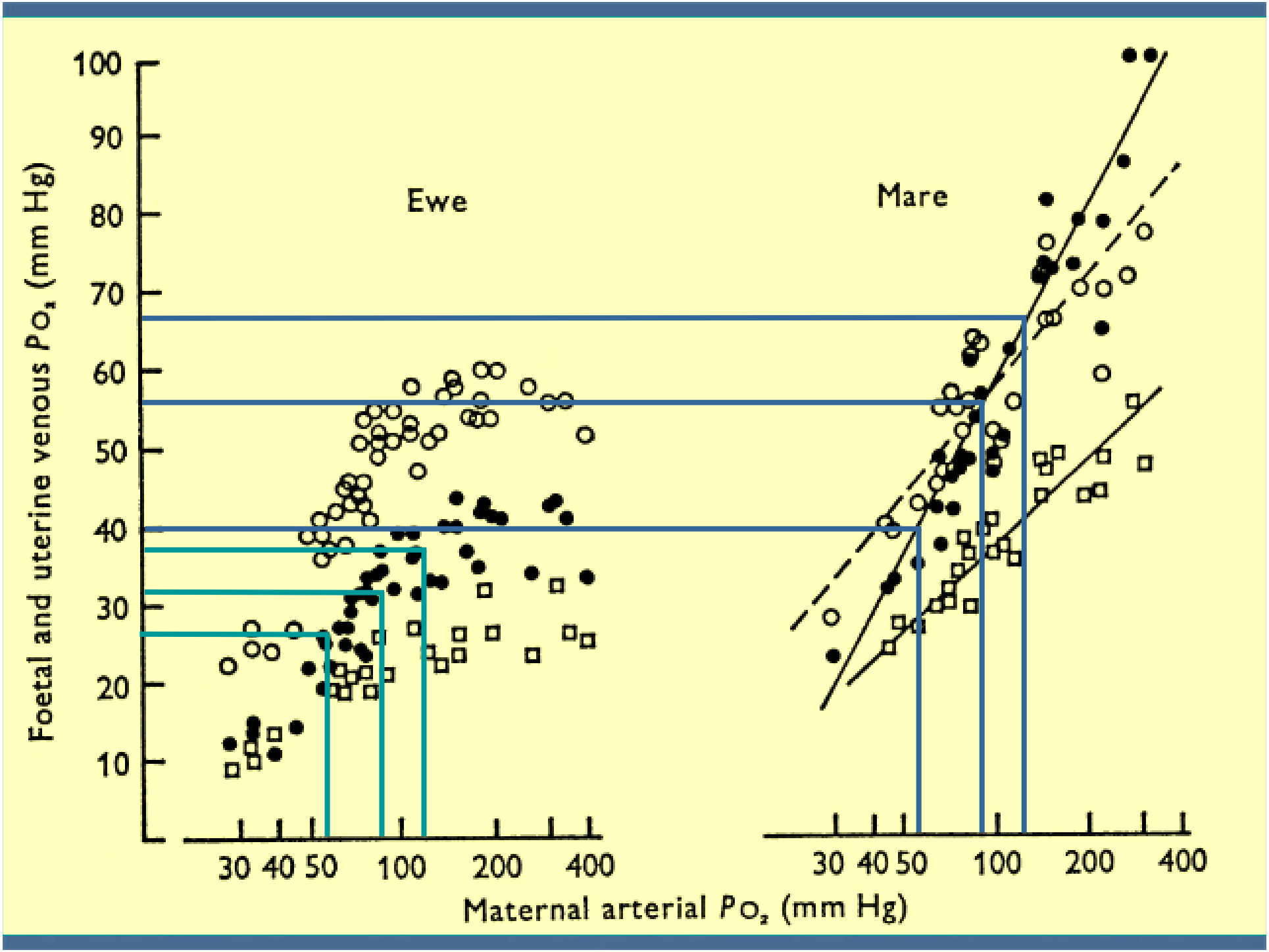


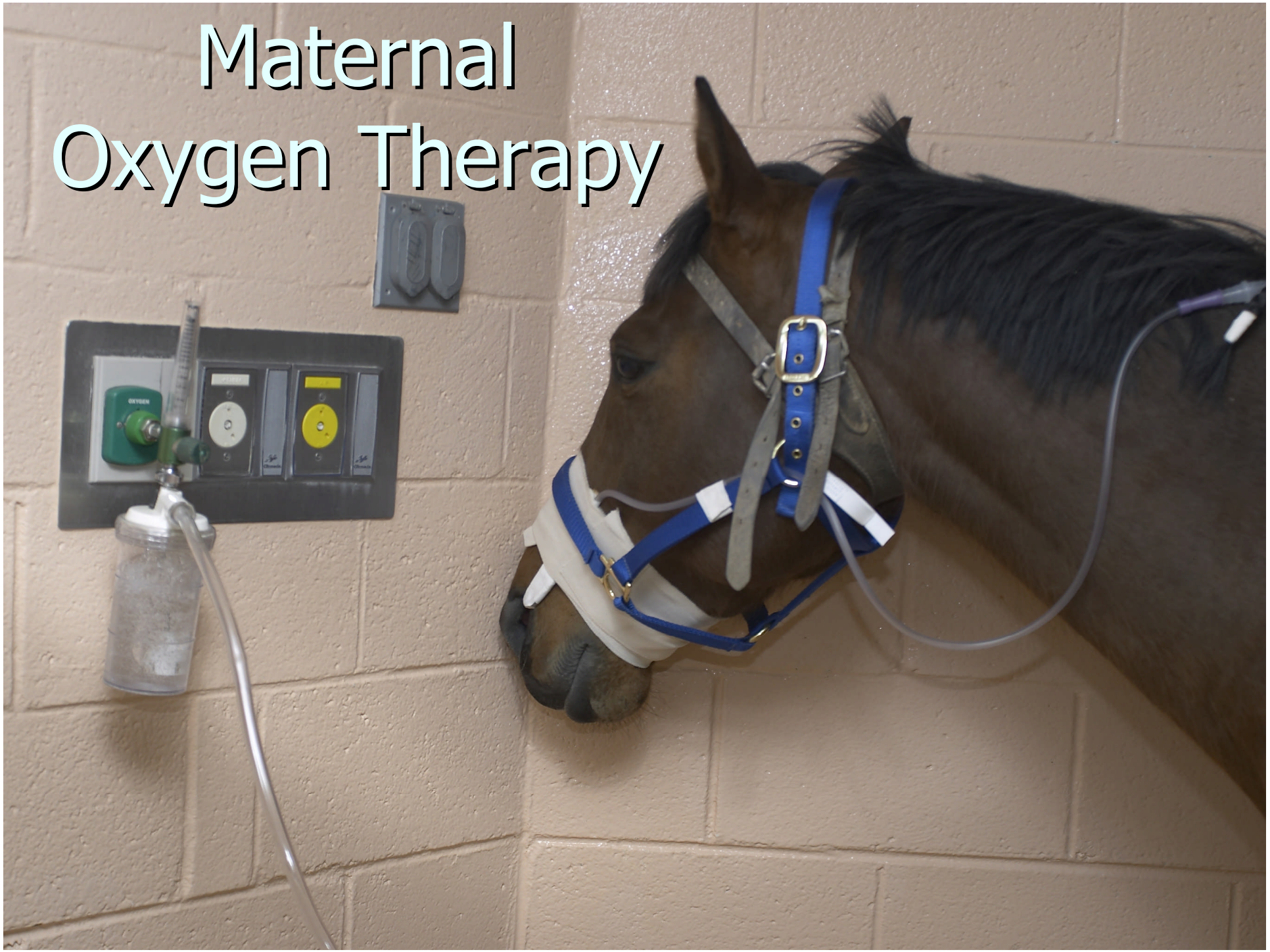
Fig. 4. The relationship between P_{O_2} in maternal arterial blood (log scale) and that in the uterine vein (○) and umbilical vein (●) in seven ewes and seven mares (data from Comline & Silver 1970*b*), and in five sows.


Ewe
Mare
Foetal and uterine venous P_{O_2} (mm Hg)
100
90
80
70
60
50
40
30
20
10
30 40 50 100 200 400
30 40 50 100 200 400
Maternal arterial P_{O_2} (mm Hg)

Effect of Placental Circulation Pattern on fetal Po_2

	Counter current circulation	Other circulation
Normal Conditions	Po_2 48-54 torr	Po_2 30-34 torr
Maternal hypoxia	↓↓↓ fetal Po_2	↓ fetal Po_2
↑ Maternal Pao_2	↑↑ fetal Po_2	↑ fetal Po_2

Maternal Oxygen Therapy



Fetal Resuscitation
Lack of O2 Delivery

- Maternal Anemia - blood transfusions
- Fetal hypoxemia - supplement with INO_2
  - Take advantage of the countercurrent system
  - Even if normal Pao_2 in mare, foal may benefit
  - Could be important with placental edema

Placental Functions
Glucose Transport

- Predominant source of energy for fetus
- Fetal:maternal glucose ratio
  - Man & Rabbits 70-80%
  - Horse 50-60%
  - Pigs 40-50%
  - Ruminants 20-30%

Placental Functions Glucose Utilization

- The placenta
  - Actively metabolic tissue
  - High glucose utilization by placenta in horse
  - Glucose for placenta also comes from fetus
- Maternal distress – less glucose
  - More glucose delivered from fetus
  - Can lead to negative net glucose transport to fetus

IUGR
Intrauterine Growth Restriction

Threats to Fetal Well-being Nutritional Threat of Acute Fasting

- Fasting the mare for 30-48 hr
  - Decreased glucose delivery
  - Rise in plasma FFA
  - Increased PG's in uterine and fetal tissues
- Increased risk of preterm delivery
  - Within one week of ending the fast
    - Associated with myometrial sensitivity to hormones
- Prevent by intravenous dextrose infusion

Fetal Resuscitation Nutritional Threats

- Support the mare's nutritional needs
  - Enteral supplementation
  - Parenteral supplementation
  - Encourage a high plain of nutrition
- Avoid acute fasting
  - Avoid elective procedures requiring fasting
  - Encourage anorexic late term mares to eat
  - Supplement with intravenous glucose therapy
- Consider flunixin meglumine therapy

Response to Hypoxia

- Hypoxia in adult
  - CO responsive to tissue O_2 levels
  - Hypoxemia results in
    - Increased CO
    - Increased gas exchange lungs
- Hypoxia in fetus
  - Hypoxemia results in
    - Decreased CO (decrease in HR)
    - Slowing placental perfusion
    - Increase BP
    - Requires intact CNS-adrenergic response

Fetal Adaptation to Hypoxia

- Unlike the lungs, placenta will not deliver more O_2 in response to fetal hypoxemia
- Increase umbilical venous resistance
  - Increases fetal placental surface area
  - Improves maternofetal gas exchange

Fetal Adaptation to Hypoxia

- Fetus/neonate - half of O_2 use is facultative
  - Growth, anabolic processes, thermoregulation
  - Not essential for survival
- Lamb
  - Fetal lamb – 30-40% of O_2 used for growth
  - Neonatal lamb – 30% of O_2 used for growth
- Induced maternal hypoxemia
  - Decrease O_2 delivery by 50%
  - No anaerobic metabolism, no fetal acidosis

Fetal Adaptation to Hypoxia

- Tolerance to acute hypoxia
  - Turn off growth – how?
  - Decrease unnecessary activity (fetal breathing)
- Tolerance to chronic hypoxia
  - Hypoxia – tolerant cells
  - Postulated to have fewer ion channels
    - Require less energy to prevent Ca leak
- Organs with high metabolic activity
  - Brain, heart
  - Smaller % O_2 used for growth
  - Need to maintain O_2 delivery in face of hypoxemia

Fetal Adaptation to Hypoxia

- Other changes
  - Decreased activity
    - Fetal activity stops
    - Fetal breathing stops
    - Fetal swallowing stops

Fetal Adaptation to Hypoxia

- Decrease CO (decrease HR)
  - Decrease cardiac O_2 need
  - But increase BP – cardiac work not change
- Redistribution of combined ventricular CO
  - To heart & brain increase from 7% to 25%
  - Increase to adrenal circulation

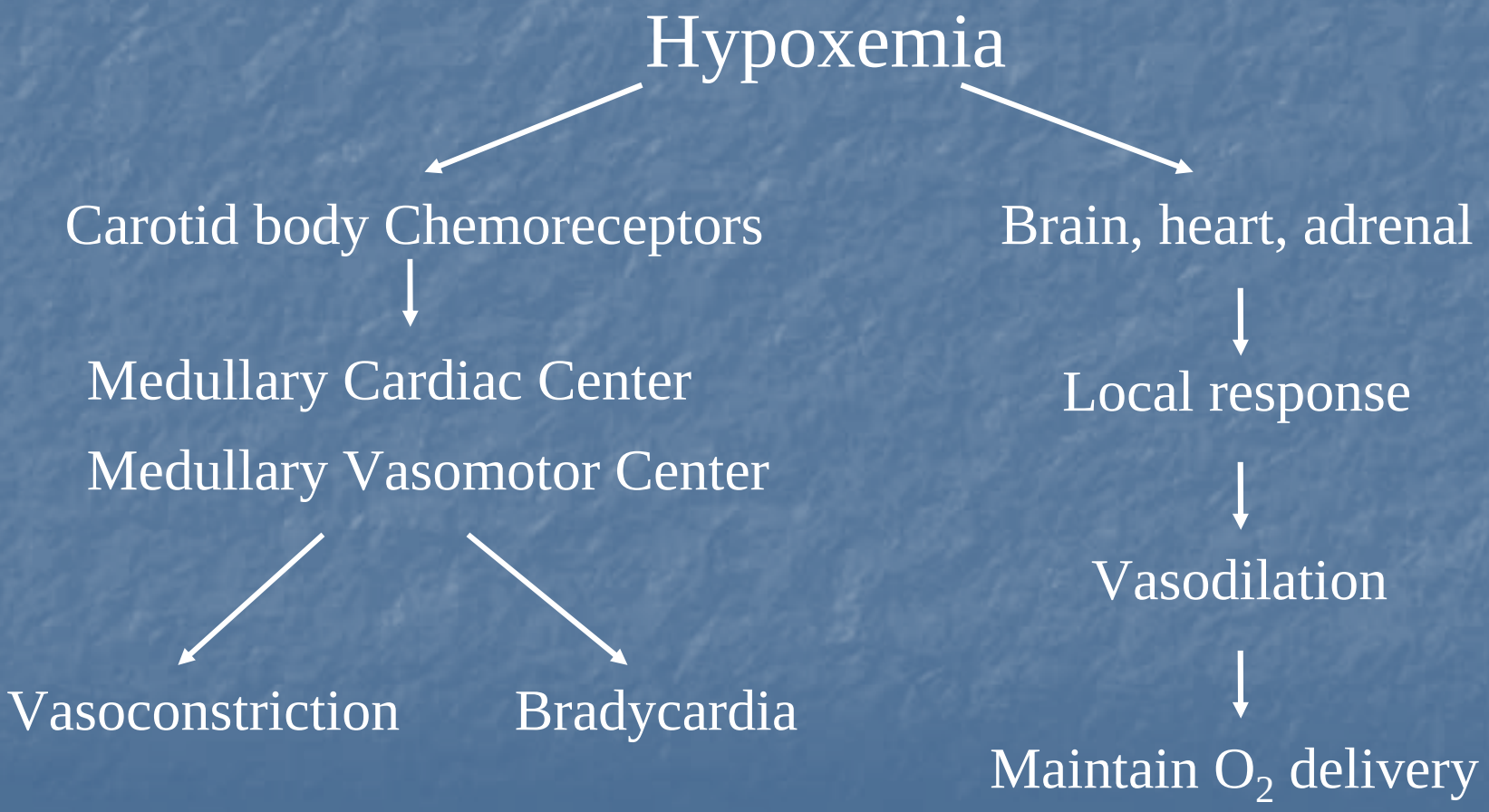
Fetal Adaptation to Hypoxia/Stress

- Maternal systemic origin
  - Less O_2/nutrients delivered from mare
- Placental or fetal origin
  - Higher demands
- Differs from adult physiology
- Maternal placenta delivery is fixed
  - Increased extraction of limited resource
  - Redistribution of limited resource

Fetal Adaptation to Hypoxia/Stress

- Increase fetal extraction
  - Increase umbilical venous resistance
    - Slow blood flow
    - Increases fetal placental surface area
- Redistribution of resources
  - Global increase vascular resistance
    - Increase BP
  - Local decrease vascular resistance
    - Cardiac
    - CNS
    - Adrenal

Fetal Response to Hypoxia



Fetal Response to Hypoxia Blood Flow Redistribution

- % distribution biventricular cardiac output
  - ↑ Gut, spleen, carcass
    Brain, heart, adrenals, placenta ↓
- Fetal lamb – Po_2 = 12-14 mmHg
  4 – 7 X ↓ myocardial flow
  No O2 delivery
  Myocardial performance sustained
    - ↑ sympathoadrenal activity
      coronary blood flow ↑

Fetal Response Hypoxemia FHR

- Hypoxia stressed fetus with compensation
  - Will have slow FHR
  - Will have fewer FHR accelerations
- Decompensation – hypoxic acidosis
  - Will loose central adrenergic response
  - Develop persistent tachycardia
  - Terminal bradycardia

Fetal Cardiac Response to Hypoxic Stress

- Effect of Hypoxia/Acidosis on Cardiac fn

  As long as adrenergic support (CNS)

  Po_2 < 15 , pH normal → normal ventricular fn

  Po_2 normal , pH < 6.8 → normal ventricular fn

  Po_2 < 25 , pH < 6.8 → ↓↓↓ ventricular fn

- Basis of hypoxic ischemic disease

Fetal/neonatal myocardium

- Resistant to hypoxic damage
  - Due to high rate of anaerobic glycolysis
  - Greater glycogen stores
  - Resistance of fetal cells to damage from lactic acidosis
    - Lactate as cardiac energy source?
- Resistance to combined hypoxia/hypercapnia
- Resistance to ischemia dysfunction
  - After short periods quickly recovers
  - Will work as efficiently as before insult
- Resistant to ischemic myocardium cell death
- More likely to survive cardiopulmonary arrest

Fetal Response to Maternal Oxygen Therapy

- Positive response
  - Increase in FHR base line
  - More FHR accelerations
- But ....
  - Most fetal foals have low FHR base lines
  - Probably reflects efficiency of CV system
  - Utilize oxygen for growth

Fetal heart rate measurements
Fetal ECG

FHR = 48-52 MHR = 60